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OMPHACOCELE *Robert G. Bremer, M.D.* 225

WOLFF-PARKINSON-WHITE SYNDROME *George J. Cohen, M.D.* 232

THE HEARING AND SPEECH CLINIC. *Norma Keitel, M.D.* 236

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OMPHALOCELE

Robert G. Brewer, M.D.

An omphalocele is a congenital weakness of the umbilical ring, with associated herniation into the base of the umbilical cord of abdominal contents covered by a peritoneal sac but not by skin. Its layers consist of amniotic membrane and peritoneum. The omphalocele gives the appearance of a delicate translucent avascular wall or membrane covering abdominal organs, most often a portion of the intestinal tract.

CASE REPORT

L. M. W., a colored girl, was born prematurely at Columbia Hospital, August 2, 1955. The patient was transferred to Children's Hospital as a ward patient, August 3, 1955, with a diagnosis of omphalocele. The history stated that the baby was apparently born without difficulty and had an essentially normal delivery.

On examination the infant was found to weigh 3 pounds, 15 ounces. She appeared essentially normal in every respect except for an omphalocele 2½ cms. in diameter which extruded through the umbilical ring with the umbilical cord. The covering of the omphalocele was of cord substance. The skin around the umbilicus was of normal color and the umbilicus appeared to be normal in every respect except for this herniation through the umbilical ring.

After consultation with the attending and associate surgeons it was decided that the contents of the hernial sac should be reduced as soon as possible. Thus, six hours after admission, the patient was taken to the operating room and, under general anesthesia, the hernial sac was opened through the cord substance, which was in a 12 o'clock position in relation to the umbilical cord structures. Within the hernial sac were 5 inches of terminal ileum, the cecum, and the entire right colon. The contained bowel appeared to be of normal color, collapsed, and was adherent to the umbilical ring. Reduction of the hernia through the umbilical ring was possible after the umbilical ring was incised in the midline in the 12 o'clock position in relation to the hernia sac. The bowel was reduced into the abdomen and the cecum was placed in the right lower quadrant but not anchored. The hernia proper was repaired with silk and the skin was closed with a subcuticular catgut suture. At the completion of the operation it was felt that the bowel wall, although showing petechial spots in several places, was not bruised to any extent by the surgical procedure.

Following operation, the patient reacted from the anesthetic promptly and was placed on "nothing by mouth" and given antibiotics, 125 mgms of streptomycin, and 75,000 units of aqueous procaine penicillin every 12 hours, intramuscularly. The baby urinated periodically in apparently adequate amounts and had five meconium stools the day following surgery. She also vomited a small amount of dark brownish fluid.

The second day following surgery, there were two meconium stools, which were less in amount than the previous day. The baby's general condition appeared to be satisfactory and she received clyses and other supportive therapy as was indicated. However, the abdomen was noted to be slightly distended and the child began vomiting increasing amounts of dark green fluid. The abdomen became more distended on the third postoperative day and there was more vomiting and no stools were passed.

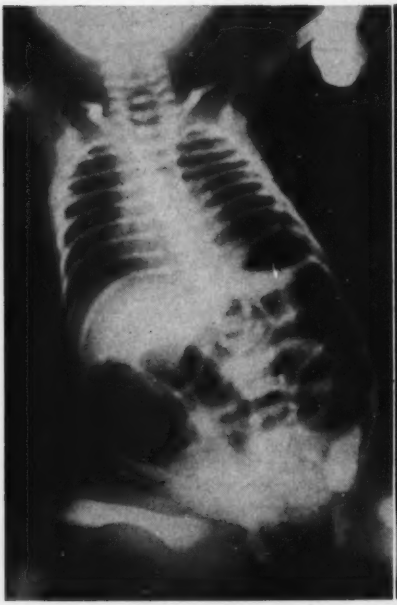

FIG. 1.

FIG. 2.

FIG. 1. August 6, 1955. Third postoperative day of omphalocele repair. Abdominal distention from markedly dilated loops of small intestine, indicating intestinal obstruction.

FIG. 2. August 8, 1955. First postoperative day after first abdominal exploration. Dilated loops of small intestine with large gastric bubble. Note large dilated loop of small intestine in right lower quadrant indicating obstruction.

X-rays at this time showed dilated loops of intestine with fluid levels indicating intestinal obstruction, rather than postoperative ileus. (Fig. 1) It was decided early on the fourth postoperative day, to re-explore the abdomen surgically through a right rectus incision to determine the cause of obstruction.

As the peritoneal cavity was entered, multiple markedly dilated loops of small intestine extruded through the incision. These loops were dilated to a diameter of approximately 2 cms. The entire small bowel was examined from the ligament of Treitz down to the terminal ileum. In the region of the terminal ileum, approximately 3 inches from the ileocecal valve, the ileum was constricted to 5 mm. and the bowel remained this diameter through the midtransverse colon. The constricted segment of bowel corresponded to that portion of bowel which comprised the contents of the omphalocele. In an attempt to correct the intestinal obstruction, a longitudinal incision was made through this constricted portion of ileum and it was closed transversely, using a Connell type of suture. The child was then returned to the ward; Wangensteen suction was instituted in an attempt to decompress the upper bowel;

and nothing by mouth and continuation of the same antibiotics were ordered. A venous "cut down" was done for fluid therapy.

Following this operation the patient's general condition remained satisfactory; she urinated adequately; and her temperature remained normal. However, the abdomen remained distended and vomiting continued, sometimes around the tube for Wangensteen suction, which appeared to be draining satisfactorily. No stools were passed. This condition persisted and more x-rays were taken which were interpreted as follows: "Numerous loops of distended small bowel with one loop of bowel in the right abdomen which is markedly distended." (Fig. 2) The x-ray diagnosis was that of a small bowel obstruction, probably in the ileum. It was decided that the baby's condition was not going to improve and that the abdominal distention and vomiting were not due to a postoperative ileus, but to an obstruction which demanded reexploration of the baby's abdomen for a second time.

On the second postoperative day following the second operation, and the sixth postoperative day from the first operation, or on the seventh day of life, the peritoneal

FIG. 3.

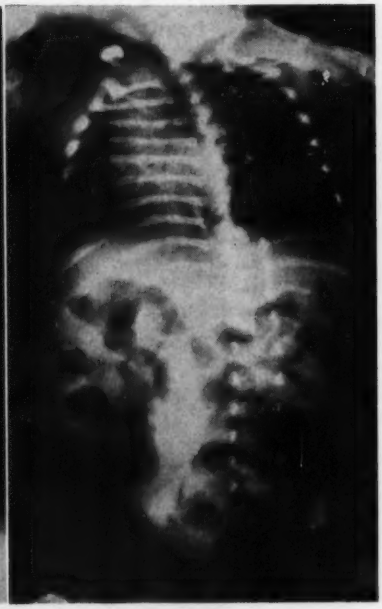

FIG. 4.

FIG. 3. August 11, 1955. Second postoperative day after second abdominal exploration. Some dilated loops of small intestine in right abdomen. No evidence of obstruction as on August 8, 1955. Note interrupted wire retention sutures in right abdomen.

FIG. 4. September 16, 1955. Approximately six weeks old. Preliminary film of abdomen shows normal gas pattern of the intestines with normal distribution. Air is in both small and large bowel.

cavity was re-entered through the right rectus incision. The small bowel was examined from the ligament of Treitz to the cecum and the large bowel was examined from the cecum to the rectosigmoid. On this examination the small bowel was less dilated than on the previous examination. However, it was still dilated to 1½ to 2 cms. The suture line in the terminal ileum was examined and was found to be closed. The section of bowel which had previously been the contents of the omphalocele appeared to be much more dilated than at the previous operation. In this examination, as during the previous operation, the intestinal contents could be pushed through this segment of the intestines, but it appeared not to be functioning properly. The large bowel distal to the point where the colon had been in the omphalocele was collapsed and appeared empty. It was decided that this segment of bowel should be bypassed, including 5 inches of terminal ileum and the entire right colon up to the hepatic flexure. A side-to-side ileo-transverse colostomy was done using 4-0 silk sutures, with a double posterior row and a single anterior row of sutures, using the Connell inverting suture. The abdomen was closed with a through and through wire suture. During the immediate postoperative period the patient was in fair condition, with a normal temperature and was voiding satisfactorily. The same antibiotic therapy was continued and fluids were given by venous "cut-down."

During twenty-four hours following this last procedure, a small amount of stool mixed with mucus was passed. The Wangensteen suction was discontinued and the

FIG. 5. September 16, 1955. Barium enema. Barium fills the rectum, sigmoid and transverse colon, traveling into ileum which courses down toward rectum in the x-ray above. Fine, string sized portion of proximal transverse colon is filled beyond anastomosis.

child continued to spit up a small amount of greenish fluid for approximately thirty hours. On the second postoperative day, there were three meconium stools. Voiding was regular and adequate in amount. The temperature remained normal or slightly subnormal, and during the second postoperative day an oral electrolyte mixture was offered to the infant; two feedings were vomited; the third feeding was retained. During the fourth postoperative day, the patient was placed on Olac, 1 tablespoon to 3 ounces of water, giving a half ounce, as tolerated, every two hours. The general condition improved, the abdominal distention decreased and there was no more vomiting. Antibiotics were discontinued on the fourth postoperative day. (Fig. 3.)

At 11 weeks of age, the baby continued to have stools daily, usually two to three in number. She voided regularly and the temperature was subnormal or normal except for one unexplained temperature elevation to 102° on the fourth day following the last operative procedure. Intravenous fluid was continued and included approximately 200 c.c. per 24 hours, one-third of this being Ringer's solution with glucose and two-thirds being glucose and water.

The only complication occurred when the left saphenous vein at the femoral junction was used for a cut-down over a period of 48 hours. The left leg suddenly became blue throughout its entire length. The cut-down was discontinued, the leg was elevated and warm soaks were applied. The leg improved somewhat immediately and over a period of 48 to 72 hours, the color returned practically to normal. On the ninth postoperative day the leg had the same diameter and color as the right leg.

On the fourth day following the last operation the infant weighed 4 pounds. Three days later, the infant weighed 4 pounds, 1 ounce, and on the ninth postoperative day the infant weighed 3 pounds, 10 ounces. This can largely be explained by the fact that the child was somewhat over-hydrated, which was necessary because the "cut-down" could not be maintained by giving the child any less fluid. At 11 weeks of age, the patient had gained only 21 ounces from birth. Some of this failure to gain may be accounted for by the occurrence of diarrhea for 4 days, during which the baby lost 4 ounces. This weight was regained and diarrhea was well controlled for a few days.

A barium enema and G. I. Series were done Sept. 16 to try to explain the cause of abdominal distention and to see whether any portion of the dormant bowel was functioning. (Fig. 4, 5) Fluoroscopic examination of the barium enema showed that the ileotransverse colostomy was patent and functioning well and only a short string sized segment of barium was seen to enter the colon proximal to the point of anastomosis.

Six to eight stools were passed daily by this infant from the last week of September until the twenty-first of October at which time the child expired rather suddenly.

An autopsy was done, which revealed the anastomosis to be patent and the terminal ileum and proximal colon to be normal in size. A precise cause of death could not be given.

DISCUSSION

This case is reported as it has some features not usually seen with the common omphalocele as reported in the literature. It was complicated by several factors. Preoperatively, the omphalocele appeared to be a typical one that was rather small in size, the diameter of the membranous portion measuring approximately 4 cms. and the diameter of the ring measuring 2½ cm. However, as the sac was entered, the omphalocele was found to

contain a portion of terminal ileum and proximal large bowel. This bowel was firmly adherent to the umbilical ring and it was with some difficulty that the bowel was released without perforation. It is well known that perforation of the bowel by forceful manipulation is an ever present possibility in pediatric surgery. In order to reduce the omphalocele, the umbilical ring was incised and enlarged to allow adequate room for reduction of the contents of the omphalocele.

Gross[1] describes 88 cases of omphalocele and does not mention adhesions as a complication of a simple repair. He does mention that in secondary repairs, no adhesions have been encountered in his series of cases. The decision as to whether to do a primary or secondary repair is made at the operating table. An estimation of the size of the abdominal cavity is made and if the contents of the omphalocele can be reduced into the abdominal cavity without causing any symptoms or complication, a primary procedure is done. If any of the hazards or complications are likely to occur, a second procedure is done at a later date, i.e., 3 months to 2 years later, depending upon when the abdominal cavity can receive the herniated contents.

The three main dangers of doing primary closures on all omphaloceles are:

1. Greatly increased abdominal pressure, resulting from the reduction of the herniated contents, causing respiratory embarrassment by elevation of the diaphragm.

2. Extreme pressure on large veins, especially vena cava, causing vascular stasis with vascular collapse.

3. The possibility, the least likely of the three, of partial intestinal obstruction due to the overcrowding of the abdominal contents.

In the review from Boston Children's Hospital, the prognosis of this condition is correlated with the size of the sac. An infant with a sac less than 7 cms. in diameter, without other serious congenital malformation, has a 75 per cent chance of survival following surgery. As the diameter of the sac increases, the chance of survival is less. A sac larger than 9 cms. reduced the chance of survival to about 15 per cent.

A second feature of this case, which makes it somewhat different from the usual omphalocele, is the fact that the bowel which was trapped in the omphalocele, when first visualized, appeared to be narrowed. On closer examination this narrowing was probably the result of chronic constriction by the umbilical ring, together with the prematurity of the infant. The bowel in the omphalocele was of a constant diameter, measuring approximately 6 to 8 mms. It was believed that this bowel was patent, thus no definitive treatment of the bowel was done. It was simply replaced into the abdominal cavity.

Gross states that 59 per cent of his cases had associated anomalies. In

the majority of these cases, the anomalies were of minor importance and did not threaten the life of the patient. He mentions anomalies of serious importance in other organ systems, but does not specifically say that he had seen any serious abnormality of the organs or bowel that were found in the omphalocele sac.

In the early postoperative period, when a diagnosis of bowel obstruction was made, we did not think that the cause of obstruction was the result of maldevelopment of that portion of the intestinal tract caught in the omphalocele. When the terminal ileum and proximal colon were examined at the first exploratory laparotomy, it was found to be lying within the abdominal cavity with the same configuration as when seen in the omphalocele. The lumen was approximately the same size, the loops were coiled in the same manner, and there was still an area which appeared to be constricted as if the umbilical ring were binding it. This probably is explained by the presence of the intestine in the omphalocele sac for a long period of fetal life, with a tight constricting umbilical ring causing improper nutrition and development of the contained bowel. In retrospect, although this bowel was patent it was not functioning and did not have peristalsis.

Gross describes the development of the coelomic cavity during the sixth to the tenth week of embryological life. Early in the development of the coelomic cavity, it is much too small to encompass the abdominal contents, but after the tenth week, the coelomic cavity, which is later the abdominal cavity, enlarges rather rapidly and soon envelops the abdominal contents. The omphalocele forms when the abdominal cavity does not enlarge enough to swallow up the abdominal contents, usually a portion of the intestinal tract and occasionally the liver. From an embryological point of view, any defect in the intestinal tract is a second congenital abnormality. In this case we believe that this defect in the closure of the abdominal cavity acted as a mechanical constricting ring catching the terminal portion of the small bowel and the proximal portion of the large bowel before it was withdrawn into the abdominal cavity, with the resultant condition described.

At the second exploratory laparotomy, it was hoped that we would find that portion of bowel that was caught in the omphalocele to be dilated to normal size and functioning normally and that the obstruction would be a constriction at the point of intestinal closure done at the first laparotomy. However, this was not the case. That portion of bowel in the omphalocele, although somewhat larger in diameter, still was much smaller in diameter than it normally should be, and it lacked any peristaltic waves after a brief observation period. Due to the patient's poor condition, it was decided to bypass this portion of the intestinal tract rather than resect it. The point of anastamosis between the dilated terminal ileum and the transverse colon

was chosen with some forethought. We did not want to bypass the dormant portion of intestine at any great distance proximal to its beginning because this would include a loop of dilated terminal ileum which could easily be the cause of perforation of the suture line.

I am not sure whether this infant died because of a mechanical derangement in the intestinal tract, or because of the introduction of infection. It is a fact that the infant had passed six to eight stools a day for a period of four weeks. During this time there was no weight gain; consequent chronic nutritional deficiency, together with an electrolyte imbalance may have been more than the child could overcome. It is also possible that the ileo-transverse colostomy created such a short segment of colon that proper absorption of fluids was not accomplished. However, if the terminal ileum and proximal colon were functioning, as they were thought to be when viewed at the post-mortem table, mal-absorption would not be a factor in this case.

SUMMARY

This is a case report of omphalocele complicated by adhesions of the intestines to the wall of the umbilical ring, and an abnormality of the contents of the omphalocele sac. These two situations are not reported occurring in Gross' description of his cases or in other cases described in the literature.

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WOLFF-PARKINSON-WHITE SYNDROME

George J. Cohen, M.D.

Although the Wolff-Parkinson-White Syndrome of short P-R interval and abnormal QRS complex appears not infrequently in adults, it is seen much less often in children. Perhaps this is because electrocardiograms are a less frequent procedure in children. Because of the relatively uncommon occurrence of this syndrome in children, the following case is presented. It is felt to be of special interest because there were two episodes of paroxysmal tachycardia with an intercurrent normal rhythm before the appearance of the Wolff-Parkinson-White pattern (hereafter designated as WPW).

CASE REPORT

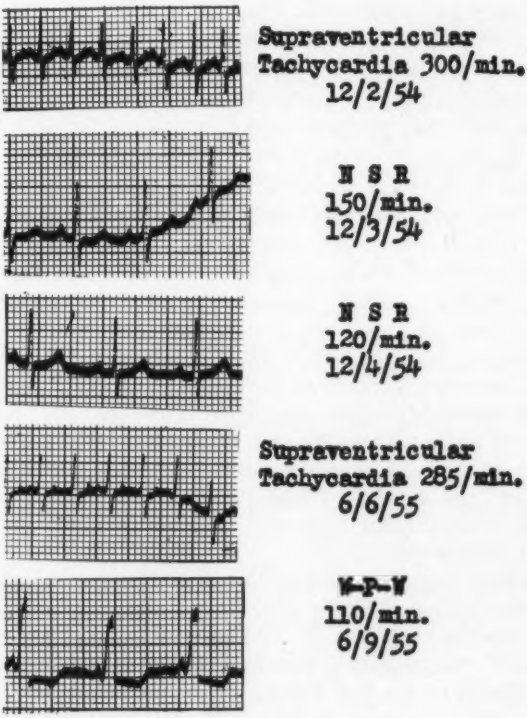
This 1½ year old colored girl was entirely well until age 10 months when she was admitted to Children's Hospital for the first time on December 2, 1954 because of vomiting and refusal to eat for two days following an upper respiratory infection of

two weeks' duration. On admission, physical examination showed a well developed and nourished negro baby in no acute distress. Her pulse and heart rate were too rapid to be counted, but seemed of regular rhythm. The cardiovascular system was otherwise normal by physical and fluoroscopic examination. Electrocardiogram (ECG) showed a regular rate of 300 per minute with slight deflections of the ST segments, indicating paroxysmal supra-ventricular tachycardia.

Immediately after admission, carotid sinus and eye ball pressure produced no observable change in heart rate, so 0.3 mg. of lanatoside C (Cedilanid) was administered intravenously. Within 12 hours the child had stopped vomiting; her pulse and apical rate were 140 to 160; and the ECG was entirely normal. She was given no more digitalis and remained without symptoms or signs of illness throughout her four day hospital stay. She was not placed on a maintenance dose of digitalis.

At home she maintained normal growth and development and remained well for six months until June 6, 1955 when she was readmitted with a three day history of vomiting, again following an upper respiratory infection of two weeks duration.

Physical examination and routine laboratory studies were normal with the following exceptions: the pulse and apical rates were approximately 250 per minute; and the liver edge was palpable one finger's breadth below the right costal margin. Chest



x-ray with barium swallow was normal. ECG revealed a supraventricular tachycardia of 280 per minute.

Eye ball and carotid sinus pressure were again unsuccessful in reducing the heart rate. Because no veins were readily entered, digalen was given intramuscularly, 75 mgm on admission and 37 mgm four and eight hours later, for a total digitalizing dose of 150 mgm (1.5 USP units). Eighteen hours later there was no vomiting, the liver edge was no longer palpable, and ECG showed a classical WPW configuration of short PR interval and wide, slurred QRS complex. On her second hospital day, a maintenance dose of 30 mg of oral digalen daily was begun.

She was discharged on June 16, 1955 after 9 days without symptoms during which ECG repeatedly demonstrated the WPW pattern. During two weeks at home she remained well and when seen again on June 30, 1955 her cardiovascular system was normal on physical examination. ECG showed persistent WPW pattern.

On September 29, 1955 she was re-examined. Growth, development, physical examination, and cardiac fluoroscopy were all entirely normal. ECG showed no change in the WPW configuration, even after subcutaneous injection of 0.12 mg of atropine sulfate. Her maintenance dose of digalen was continued.

DISCUSSION

The electrocardiographic pattern of a short PR interval with abnormal QRS complex was first described in 1930 by Wolff, Parkinson, and White, who considered it a functional bundle branch block with shortened atrio-ventricular conduction time due to vagal influence[1]. This ECG finding was observed by them primarily in healthy young adults who also had episodes of paroxysmal tachycardia. In their original series of 11 patients there was no mortality or significant morbidity.

In 1948 Wolff and White reviewed the literature which had developed since their original article, and compiled data on 52 cases including the first 11[2]. In this study it was found that the electrocardiogram of some patients manifested abnormal ventricular complexes even during tachycardia. While some electrocardiograms showed reversion to normal patterns spontaneously as well as on medication, others remained abnormal in spite of digitalis, quinidine, atropine, and vagal stimulation. It was postulated that an accessory atrio-ventricular conduction pathway produced the syndrome, and this hypothesis has been concurred in by subsequent authors. Histological evidence of aberrant muscle bundles between auricle and ventricule has been shown by Wood, et al[3] and by Kimball and Burch[4] on autopsy material from patients with known WPW who died with paroxysmal tachycardia.

Recent articles have reported that this anomaly can occur in infants, and can be incapacitating and even fatal.[5] [6] [7] Engle in 1952 carefully reviewed the literature and pointed out that in the majority of infants with this syndrome, the presenting picture was congestive failure with paroxysmal tachycardia with subsequent absence or delay in specific therapy.[8]

Although there is general agreement that the tachycardia and WPW

patterns may appear and disappear spontaneously, most previous cases presented a sequence of tachycardia, WPW, normal pattern. In our case, however, only the second episode of tachycardia reverted on digitalis to a WPW configuration which has persisted for 3 months in spite of a trial of atropine and continuous digitalis maintenance. This would suggest that the accessory pathway from atrium to ventricle does not operate constantly, but may be called into play intermittently.

On both occasions our patient presented with symptoms of incipient congestive failure which responded promptly after digitalis therapy. Our own experience as well as that of many other authors would indicate that rapid digitalization is the therapy of choice in paroxysmal supraventricular tachycardia, especially in the presence of failure. The dosage should be at least 1 USP unit per 10 pounds, with slightly higher dosages for small infants. Vagal stimulation is usually an unsuccessful therapeutic technique in infants, but should be tried because of the simplicity of applying eyeball or carotid sinus pressure. When the above mentioned methods do not stop the tachycardia, or when there is ventricular arrhythmia, quinidine should be used in dosage of 3 mg per pound every 3 hours until therapeutic effect or toxic symptoms appear. Occasionally, the only effective therapy is pharmacologic vagal stimulation with acetyl-beta-methyl choline in doses of 0.5 to 1.0 mg. If there has been more than one episode of tachycardia, maintenance therapy with digitalis or quinidine should definitely be instituted. There is certainly reason to believe that even one attack of tachycardia in a patient with WPW syndrome warrants prolonged maintenance treatment.

SUMMARY AND CONCLUSIONS

A case of WPW syndrome has been presented in which an initial episode of tachycardia reverted to a normal pattern while a second episode converted to a continuing WPW pattern, in spite of maintenance digitalis and a trial of atropine.

The cause of this syndrome would seem to be an accessory A-V conduction pathway.

In infants with WPW syndrome the occurrence of congestive failure and death indicates the potential seriousness associated with this conduction anomaly.

Digitalis administration is usually the treatment of choice of the associated paroxysmal tachycardia, and should be maintained to prevent recurrence of tachycardia.

ACKNOWLEDGMENT

The author wishes to thank Dr. John O. Nestor for invaluable help in managing the patient and in preparing this paper.

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THE HEARING AND SPEECH CLINIC

Special Report

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Speech is the most complex of all human accomplishments and it is therefore not surprising that there are an estimated 4,000,000 speech handicapped individuals in this country. Although a clear cut distinction between normal and abnormal speech cannot always be made, Van Riper's definition of a speech defect is perhaps the most helpful: speech is defective when it deviates so far from the speech of other people that it calls attention to itself, interferes with communication or causes its possessor to be maladjusted.

Most speech disorders are noted during the preschool years at a time when acquisition of language is most dramatic and when attention of family and physician is most likely to be focused upon its development. The important job of prevention of the serious consequences of these disorders should be undertaken at this time, for the emotional, social and academic problems imposed upon children with language disabilities are severe.

The frequency of occurrence of the different types of speech defect as

reported in the White House Conference (1931) correlates with the experience in the speech clinic at Children's Hospital:

(A)	Articulatory disorders	72%
	(sound substitutions)	46%
	(organically caused)	8%
	(oral inactivity)	11%
	(dialect)	5%
(B)	Rhythm disorders	22%
(C)	Voice disorders	4%
(D)	Others	2%

Disorders of articulation include those characterized by sound substitutions, omissions, and elisions, the familiar infantile perseverations, lisping and delayed speech. The most common defects whose etiology is organic are those associated with hearing impairment, mental deficiency, cleft palate, cerebral palsy and the aphasias. The disorders of rhythm include most frequently stuttering, stammering, cluttering and spastic speech. Disorders of phonation include those characterized by abnormalities of voice quality, pitch, and intensity and are seen most commonly in our experience in the children with hearing impairment, cleft palate or abnormal adenoidal growths.

A challenging group of problems which interest us particularly in the clinic at the present time are the dysphasias, which include all the disorders of symbolization. They are frequently seen in milder form as specific disabilities in one or more components of the language complex: speech, reading and spelling. Children in this group present very definite academic problems requiring remedial care. Less well understood is severe congenital aphasia which must always be considered in the differential diagnosis of mental deficiency, hearing impairment and emotional disturbance in a non-talking child.

Diagnostic facilities offered by the Children's Hospital Hearing and Speech Clinic under the direction of Dr. Allen E. Walker, include otological examinations, standard audiometric procedures as well as the psychogalvanic skin resistance (PGSR) test. The PGSR was developed at Johns Hopkins University School of Medicine to obtain threshold audiometric measurements with infants and very young children. It has proved to be a valuable adjunct to the differential diagnosis of hearing impairment, emotional disturbance, dysphasia and amentia in children too young to respond to standard procedures.

Children whose speech defect is the result of hearing impairment are accepted for language training with Mrs. Edna K. Monsees, Audiologist.

Those children whose language problems are not associated with hearing

impairment are referred to the Speech Clinic for evaluation, further diagnostic work-up and therapy.

Language disabilities are encountered by all physicians dealing with young children. Any child who is not talking or responding normally to sound by the age of 2 or any child whose speech continues to be severely defective at the age of 3 years should have audiometric and speech evaluation. Although language problems are sometimes "outgrown" it is important to recognize them at a time when therapy and reassurance of the family will be most effective in the prevention of subsequent emotional, social and academic maladjustment.

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